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The high sustained $+G_z$ acceleration of aerial combat maneuvering represents a unique cardiovascular stress. Twenty-two healthly males were subjected to exhaustive simulated aerial combat maneuvers (SACM) on a centrifuge, and the time course of the P, QRS, and T-wave voltage changes was followed. Marked changes in the T-wave were noted both during and after SACM. Characteristically T-waves were tall, peaked, with a narrow base; the maximum mean change in T-wave amplitude was 5.1mv at 1 min. post SACM. Although the individual
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R and S-waves changed significantly, the overall QRS amplitude did not change. The normal response of apparently healthly asymptomatic individuals to maximum exhaustive G-stress includes a decrease in R-wave amplitude similar to treadmill exercise response seen in healthly individuals. The etiology of the electrocardiographic changes to $+G_z$ stress has not been fully elucidated. Serum potassium levels were increased immediately post SACM as compared to pre-G_z stress levels. Whether or not hyperkalemia significantly contributes to the late and post SACM T-wave amplitude and configurational changes is unknown. Adrenergic stimulation and other changes undoubtedly also play a significant role. Documentation of normal electrocardiographic response to $+G_z$ stress and correlation with known hemodynamic changes resulting from $+G_z$ stress should increase the knowledge of what factors influence the surface electrocardiogram.

Acceleration Induced Voltage Variations in the Electrocardiogram During Exhaustive Simulated Aerial Combat Maneuvering

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The high sustained +G_z acceleration of aerial combat maneuvering represents a unique cardiovascular stress. Twenty-two healthy males were subjected to exhaustive simulated aerial combat maneuvers (SACM) on a centrifuge, followed by the time course of the P, QRS, and T-wave voltage changes. Marked changes in the T-wave were noted both during and after SACM. In addition to characteristically being tall peaked T-waves with a narrow base, the maximum mean change in T-wave amplitude was 5.1 mV at 1 min post SACM. Although significant changes in the individual R and S-waves occurred, the overall QRS amplitude did not change. The normal response of apparently healthy asymptomatic individuals to maximum exhaustive G-stress includes a decrease in R-wave amplitude similar to the response to treadmill exercise seen in healthy individuals. The etiology of the electrocardiographic changes to +G_z stress has not been fully elucidated. Serum potassium levels were measured and found to be increased immediately post SACM as compared to pre-G_z stress levels. Whether or not hyperkalemia significantly contributes to the late and post SACM T-wave amplitude and configurational changes is unknown. Adrenergic stimulation and other changes undoubtedly also play a significant role in the observed changes. Documentation of the normal electrocardiographic response to +G_z stress and correlation with the known hemodynamic changes resulting from +G_z stress should increase the knowledge of what factors influence the surface electrocardiogram.

VOLTAGE VARIATIONS of the electrocardiogram are well known to occur as a result of strenuous exercise. The changes have been noted to occur both during stress and in the recovery period. Changes in the T-wave amplitude and morphology have long been utilized in clinical treadmill exercise testing, with specific T-wave changes during and post-exercise being related to the presence of cardiovascular disease. Recent emphasis has been placed on the QRS complex as an aid in enhancing the diagnostic interpretation of treadmill exercise tests. These electrocardiographic changes have been related to the state of aerobic conditioning, humoral and electrolyte disturbances, changes in intracavitary (ventricular) blood volume, and other factors (psychiatric and cerebrovascular).

High sustained +G, (head to foot) acceleration is extremely fatiguing and provides a unique physiologic stress. Knowledge of the effects of +G_z stress on the surface electrocardiogram adds insight to more fully understanding the factors that influence cardiac electrophysiology. Although previous investigations have focused on the electrocardiographic response to +G, stress (3,7,10,11,12,32), this report describes the effects of exhaustive simulated aerial combat +G, stress on the amplitudes of the P, QRS, and T waveforms as seen on the surface electrocardiogram.

MATERIALS AND METHODS

Twenty-two healthy male members of the USAF School of Aerospace Medicine (USAFSAM) centrifuge acceleration stress panel were exposed to an exhaustive simulated aerial combat maneuver (SACM) +G, stress profile. The acceleration profile alternates +4.5 G, for 15 s with +7 G, for 15 s until the subject is exhausted. The rate of onset of the +G, stress was 1G/s. All subjects wore standard USAF anti-G suits. The SACM exposure was terminated when the subject was exhausted or when he could not maintain peripheral vision as previously described (8,9). These individuals were fully trained in the optimum methods of performing M-1 or L-1 straining maneuvers used to enhance +G, tolerance (23). All subjects had successfully passed a USAF Class II flying physical examination. In addition they were required

The research reported in this report was performed by members of the Crew Technology Division of the USAF School of Aerospace Medicine, Brooks AFB, Texas.

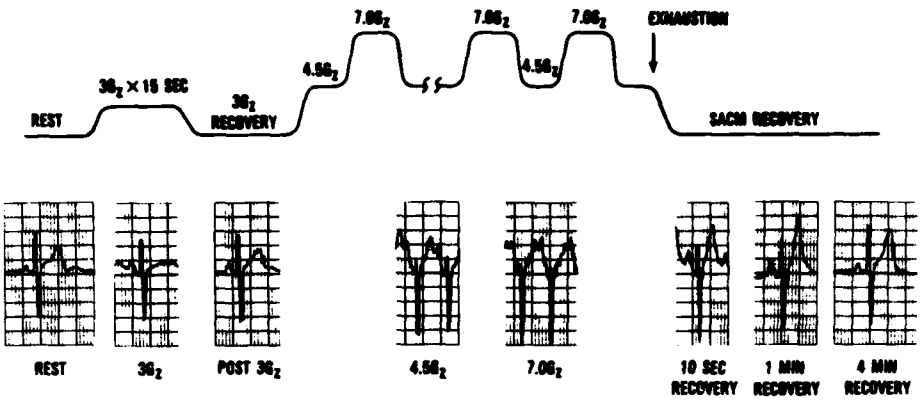

Fig. 1. Representative electrocardiographic response at specific intervals during and after exhaustive +G_z stress.

to have a normal maximal treadmill exercise test using the USAFSAM protocol (speed 3.3 mph with 5% grade increased for 3 min of successive exercise).

Each subject was instrumented with sternal and biaxillary electrocardiographic leads (32). Measurements were made from the biaxillary lead which is a modified V_1-like lead. Continuous recordings were obtained throughout the +G_z stress exposure. A representative response is shown in Fig. 1. Electrocardiographic measurements were made at rest, during a +3 G_z for 15 s warm-up exposure, and post +G_z at 10 s and 1 min. Measurements were then made immediately prior to the SACM, during the SACM in the last complete 15 s +4.5 G_z peak, and in the last complete 15 s +7 G_z peak, and post SACM at 10 s, 1 min, 2 min, 3 min, 4 min, and 5 min. The electrocardiographic measurements included the P-wave, R-wave, S-wave, overall QRS complex, and T-wave amplitudes. Each voltage measurement, in mV, was the average of the beats over a 10 s period.

Serum potassium levels were drawn from the antecubital vein of six individuals before +G_z-stress and within 1 min immediately after the SACM exposure. Potassium analysis was performed using standard flame photometric techniques. For each electrocardiographic voltage measurement, a general statistical test for time period differences was performed using a two-way analysis of variance. These were followed by specific comparisons of each time period against rest using the LSD procedure. Serum potassium levels were compared using Student's paired *t*-tests.

RESULTS

The 22 subjects in this study were all males with an average age of 25 yrs. (max age 38 yrs., min. age 18 yrs.), a mean height of 70 in (178 cm), and a mean weight of 169 lb (77 kg). Of the subjects, 11 had strenuous daily exercise programs in addition to being centrifuge acceleration stress panel members. An additional 7 subjects exercised at least three times per week and the remaining four subjects had sporadic exercise programs. The SACM duration and heart rate response to +G_z acceleration stress are given in Table I. The mean voltage amplitudes for the several periods are plotted in Fig 2 and given in Table II. The T-wave was found to show the most profound changes during and after the acceleration stress period. During the brief exposure to the low +G_z stress the T-wave showed a mild but significant decrease in amplitude. It then increased slightly post stress before returning to normal prior to the SACM. The decreased T-wave amplitude noted during the 3 G warm-up was also noted in the early phase of the SACM. As exhaustion became eminent the T-wave was significantly increased and continued to increase to a maximum in the post stress 1-2 min recovery period. Complete recovery of the T-wave to the pre-stress level had not been achieved even at 5 min of recovery. The total magnitude of the QRS complex did not change significantly except for a mild decrease during the +3 G_z stress period. Although the overall QRS magnitude showed little change, the separate R and S waves did change, with the R-wave significantly decreasing during +G_z stress. Both waves quickly returned to normal early in recovery. Although P-wave measurements and changes were made where possible, at the higher heart rates the P-waves merged into the T-waves. P-wave amplitude did show significant increases during +G_z stress and early recovery. Anticipatory changes were noted in the P, R, and S wave amplitudes prior to the severely stressful SACM.

TABLE I. STRESS DURATION AND HEART RATE RESPONSE TO +G_z ACCELERATION.

	HEART RATE (bpm)			DURATION
	REST	+3 G	SACM	SACM (sec.)
MEAN	86	124	175	186
± 1 S.D.	20	20	18	113
MAXIMUM	125	158	200	405
MINIMUM	56	80	135	46

The heart rates achieved during the SACM were equal to the maximal heart rate during maximal treadmill exercise testing performed during qualification for the centrifuge acceleration stress panel. The usual heart rate response to SACM +G, stress includes a relatively rapid stabilization to a near maximal rate with a relatively slow rise thereafter to a maximal rate at exhaustion. When the +G, stress is removed there routinely is an initial marked sinus slowing followed by a one to five minute period of marked sinus arrhythmia.

Serum potassium measurements prior to +G_z stress revealed a mean of 3.46 mg% (±0.5) with an increase to 3.77 mg% (±0.7) within the first minutes post +G_z. The change, although small was significant at the $p<0.05$ level. This amount of change is not necessarily the maximum change but simply serves to demonstrate that

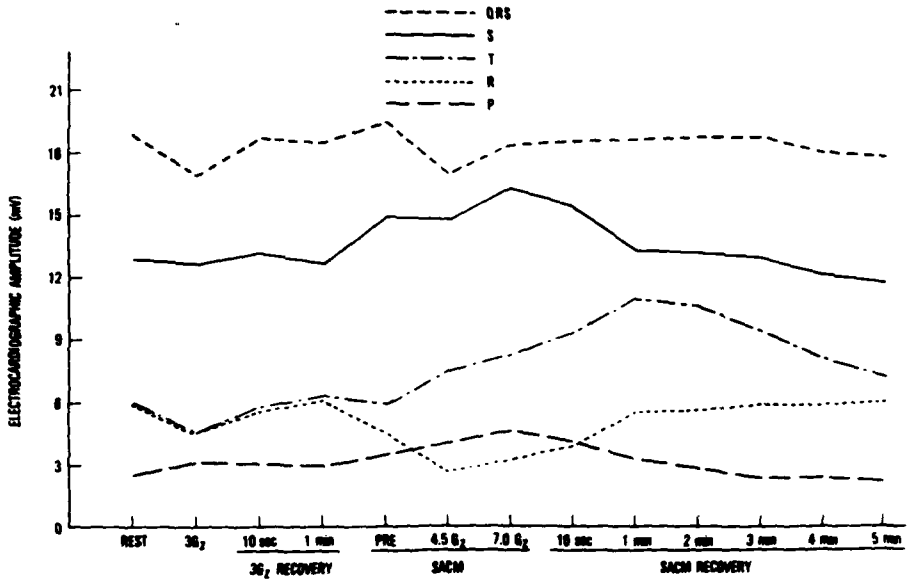

Fig. 2. Mean voltage values for the electrocardiographic parameters measured as a function of time.

potassium does increase after exhaustive $+G_z$ stress. More complete kinetic studies would be necessary to relate to the exact time-dependent rate of change of the electrocardiographic parameters with the electrolyte changes.

DISCUSSION

The electrocardiographic response to exercise has long interested both physiologists and clinicians for a multitude of reasons. Einthoven's early observations on the postexercise electrocardiogram revealed increased amplitude of P and T waves in addition to ST segment changes (13). Since that time use of the various electrocardiographic amplitude and segment changes have been related to age, electrolyte changes, aerobic conditioning, cardiovascular and other disease states, and normal accommodation to physiologic stress. The basic biochemical and physiologic mechanisms which result in the net changes observed on the surface electrocardiogram still remain to be fully elucidated. Acceleration represents a unique stress, and perturbates the cardiovascular system differently from other forms of exercise. Relating electrocardiographic amplitude changes with hemodynamic changes during acceleration to the same parameters during other forms of stress testing may help answer questions regarding the mechanisms responsible for electrocardiographic amplitude changes.

T-wave response to exercise and training

Kentala reported that adaptation of the heart to endurance athletic exercise was characterized by an increase in QRS amplitude at rest (20,21). Mayhew reported no change in the QRS amplitude but did find an increase in T-wave amplitude with training (24). Dunn found a correlation between the time for the post exercise T-wave to return (decrease) to pre-exercise heights and the accumulated time in running several 440 yard distances, and was therefore able to differentiate between superior and inferior trackmen (27,28). T-waves have been found to increase in amplitude following exercise in well trained middle aged men; and when the trained individuals were compared to non-trained runners, the T-waves were higher at rest, during exercise, and in recovery in the trained individuals (17,18). Joseph found the T-wave amplitude to decrease during treadmill exercise, with an increase during the first minute of recovery, followed by a slow decrease over the next 4 minutes to an amplitude less than the resting height (19). Rose also found peaking and increased amplitude of the T-wave in the recovery period and considered that the time required for the T-wave to return to basal levels post exercise was a function of cardiopulmonary efficiency and that a rapid rate of return was characteristic of a fully trained athlete. Ellestad suggested that the exercise induced changes in T-wave amplitude in trained individuals and young adults soon after exercise was due to an increase in stroke volume corresponding to an increased rate of decline in heart rate after exercise (14). Simoons observed both P and T-wave changes during exercise (33). He found an increased P-wave amplitude during exercise consistent with a pattern of right atrial overload. The T-wave amplitude decreased during exercise, but both

TABLE II. MEANS ACROSS TIME FOR EACH ELECTROCARDIOGRAPHIC AMPLITUDE.

EKG		During	Post 3G		Pre	During SACM		Post SACM					
Amplitude	Rest	3 G	10" post	1' post	SACM	@4.5 G	@6.0 G	10" post	1' post	2' post	3' post	4' post	5' post
P	2.5	3.1	3.0	2.9	3.5	4.4	5.1	4.3	3.2	2.8	2.4	2.4	2.2
QRS	18.9	16.9	18.7	18.5	19.5	17.1	18.4	18.6	18.7	18.8	18.9	18.2	18.0
R	6.0	4.4	5.5	5.9	4.5	2.1	1.6	3.2	5.3	5.6	5.9	5.9	6.1
S	12.9	12.6	13.2	12.7	15.0	14.9	16.4	15.5	13.4	13.3	13.1	12.3	11.9
T	5.9	4.5	5.8	6.3	5.9	7.5	8.3	9.4	11.0	10.7	9.5	8.2	7.3

P and T-waves markedly increased in magnitude in the first minute of recovery with a subsequent gradual return thereafter to normal. The decreased T-wave amplitude during exercise was consistent with a reduction of left ventricular end systolic volume, followed by it's return to the resting level postexercise after a temporary overshoot in the immediate postexercise period.

Hyperkalemia as an etiology for T-wave changes

Tall, peaked T-waves have also been seen in association with marked bradycardia, psychiatric disorders, cerebrovascular accidents, left ventricular diastolic overload, and subendocardial ischemia. A number of mechanisms have been put forth to account for post exercise increased T-wave amplitude, including positional changes, changes in sympathetic tone, increased ventricular volumes, global myocardial ischemia, and hyperkalemia (34). Hyperkalemia can cause peaked T-waves similar in shape to the post exercise T-waves. These tall peaked T-waves with a narrow base have been termed "tented T-waves" (16). It is known that potassium is increased both during exercise and for a short time post exercise. There is therefore, an apparent anomalie n considering hyperkalemia as the sole mechanism for increased T-wave amplitude since T-waves are normally known to be decreased during exercise (34). The post exercise increase in circulating potassium is transient, lasting 2 to 3 min. at most (29). Peaking and increased amplitude of the T-wave has been thought to occur after a rapid rate of change of potassium (29). Acidosis, muscular ischemia, and hemolysis all tend to increase potassium (29). An increase in potassium has previously been shown to result in a decrease of upstroke velocity and both a decrease in amplitude and duration of the action potential, along with a decrease in the rate of rise of phase 0. The T-wave changes resulting from increased potassium have been thought to be due to altered conduction velocity or primary changes in the duration of repolarization. If the configuration and duration of the QRS on the surface electrocardiogram remains unchanged, then the change in height and configuration of the T-wave is primary and indicative of accelerated repolarization (16). Other electrocardiographic changes previously observed as a result of increased potassium include a decreased amplitude and duration of the P-wave, decreased R-wave amplitude with increased depth of the S-wave, and ST-segment alterations. The rate of increase in potassium may be equally as important as the absolute magnitude of the potassium. Rhythm disturbances such as ventricular tachycardia and ventricular fibrillation have been noted with rapidly increased potassium, whereas asystole has been observed with a slow increase in potassium (16). There is not complete agreement on the resultant effects of the rate of increase in potassium (15) on electrocardiographic rhythm or voltages.

T-wave response to +G_z stress

The T-wave response to exhaustive SACM +G_z stress revealed an initial decrease in amplitude during low G-stress (less than 4.5 G, on a 1 G/s onset run), an increase during late SACM (near exhaustion), a very marked increase immediately post SACM G-stress (maximum at 1 min post stress), and a slow return toward normal thereafter (not fully recovered at 5 min post stress). As with other forms of muscular exercise, potassium levels were increased in the immediate postexercise period. The exact time course of potassium fluctuation during and post +G, stress is not known. Whether or not this transient increase in serum potassium is responsible for, or additive to other mechanisms resulting in the marked T-wave amplitude increase cannot positively be stated. The changes are certainly in the right direction. The absolute magnitude of the rise in potassium was not great. The magnitude may not be the only factor to consider since the rate of rise of the serum potassium can be also considered important. During +G_z stress, marked pooling of blood occurs in the extremities. The anti-G suit constricts the abdomen and lower extremities throughout the stress. Immediately post G stress, the anti-G suit deflates and a large increase in venous return occurs. This venous return not only increases cardiac blood volume but also should be rich in metabolites and potassium stagnated in the dependent areas during +G, stress. This rapid return of volume and increased potassium could synergistically be responsible for the resultant T-wave increases.

Catecholamines as an etiology for T-wave changes

Out results are essentially in agreement with those of Shubrooks who reported a decrease (sometimes with flattening, inversion, or becoming biphasic) in T-wave amplitude early during G, stress. The early decrease was reported to return to normal in the latter half of the G, stress runs with an increase in T-wave amplitude following deceleration (32). Catecholamines have been reported to have a marked influence on the T-wave in normal subjects (22). The early flattening of the T-wave during +G, stress followed later by an increased T-wave may represent the differential response to epinephrine and norepinephrine, respectively. Although the complete kinetics and mechanism have not been fully investigated, epinephrine and norephinephrine have been found to be markedly elevated in response to +G_z stress. From the electrocardiographic findings reported here one would predict an early rise of epinephrine leading to the observed decreased T-wave amplitude followed later by an increase in norepinephrine leading to an increased T-wave amplitude. It would however be surprising if the hormonal response were solely responsible for all of the observed changes.

QRS response to exercise and clinical considerations

More recently the QRS complex, specifically the R-wave, has clinically begun to receive more attention. An exercise induced increase in R-wave amplitude has been reported to reflect severe left ventricular dysfunction and coronary obstruction, while a decrease in R-wave amplitude has been considered consistent with normal left ventricular function (2,4). The increased R-wave amplitude was considered to be due to an increased ventricular systolic and diastolic volume and pressure. Using an R-wave summation in 8 unipolar limb and chest lead, a good correlation was found with left ventricular ejection fraction and augmented ejection fraction (1). An increase in R-wave amplitude and decrease in

S-wave amplitude have been found to be the earliest finding in acute myocardial infarction (25), coronary ligation, and variant angina (31). Adrenergic stimulation has been suggested to be responsible for the decrease in R-wave magnitude, and during maximum treadmill exercise adrenergic stimulation was shown to produce a decrease in R-wave magnitude. The decrease was additionally shown to be abolished with beta-blockage (5). These findings have not been universally accepted, as other reports have found that R-wave amplitude changes during exercise testing have little diagnostic value and are not related to exercise-induced changes in left ventricular function or volumes (2). Mathematical models have been constructed to relate to the effect of cardiac dilation and hypertrophy on the surface potential (30). The results show that both an increase in volume and an increase in mass result in augmentation of the surface potential, as suggested by Brody (6). More recently evidence has suggested that the effects of intracavitary blood volume and cardiac mass are not the only factors in the genesis of QRS changes during exercise, and neither are augmented atrial repolarization forces during exercise solely responsible for the observed QRS changes (26).

QRS response to +G_z stress

The normal response of the QRS complex to exhaustive SACM +G_z-stress is a simultaneous decrease in the R-wave amplitude and increase in the depth of the S-wave. The overall QRS amplitude stays relatively unchanged. The QRS changes may be more related to positional changes during stress than other factors. The downward +G_z forces (head to foot), in addition to the upward forces caused by inflation of the abdominal bladder of the anti-G_z suit, effectively compress the cardiac structure. Certainly during +G_z stress there are decreased end diastolic and end systolic volumes as compared to rest, with a large corrective overshoot post-+G_z stress. End diastolic and end systolic pressure have been shown to be increased both during and post +G_z stress in miniature swine (John Burns, personal communication). As previously discussed, catecholamines and potassium increase in response to +G_z stress. Which of these mechanisms—position, volume, catecholamine, electrolyte, or pressure—has the major influence on QRS amplitude and configuration is not known.

CONCLUSION

Marked changes in the electrocardiographic amplitudes do occur as a result of exhaustive SACM +G_z stress. Positional, pressure/volume, humoral, and electrolyte changes may all be significant causative factors either alone or in combination. Documentation of the normal response to this severely stressful environment should allow more accurate use of the electrocardiographic response to +G_z stress as a diagnostic screening tool in aviation medicine.

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